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Chapter 12: Wound Debridement

Dot Weir; Pamela Scarborough

INTRODUCTION

CHAPTER OBJECTIVES

At the end of this chapter, the learner will be able to:

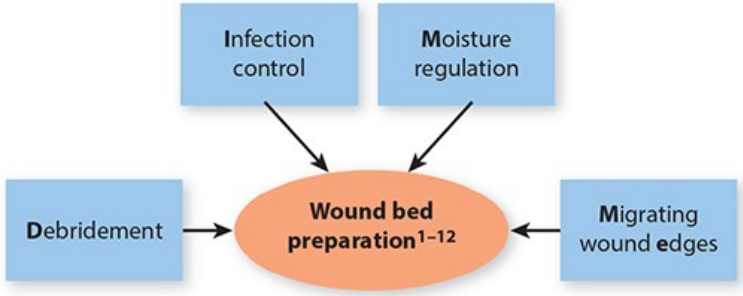
1. **Discuss the contribution of debridement to wound bed preparation.**
2. **Define the types of debridement currently used in wound care practice.**
3. **Differentiate selective from nonselective debridement.**
4. **Select the appropriate type of debridement for patients whose wounds would benefit from the procedure.**

Debridement as a Component of Wound Bed Preparation

Wound bed preparation is the global management of chronic wounds, which includes the goals of removing necrotic tissue from the wound, reducing the wound bacterial burden, managing the moisture content, and ensuring that the wound edges are conducive to cellular migration for resurfacing the wound (**FIGURE 12-1**). These components of wound bed preparation contribute to the correction of abnormal wound repair with the goal of facilitating the process of wound closure and subsequent healing. Adequate wound bed preparation is required prior to the use of advanced wound care therapies, such as cellular and tissue-based products, other biological wound interventions, collagen, and negative pressure wound therapy.[1]

FIGURE 12-1

The components of wound bed preparation are expressed in this paradigm of DIME, and can guide the clinician in the principles to facilitate wound healing.



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Debridement of necrotic tissue from a wound bed is a key component of preparing a chronic wound for closure and subsequent healing (**FIGURE 12-2**).[2–12] Numerous evidence-based wound care guidelines describe the importance of adequate and repeated debridement as a fundamental intervention for chronic non-healing wounds. One of these well-known international documents is the European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Ulcer Advisory Panel (NPUAP) guideline *Pressure Ulcer Prevention and Treatment: Clinical Practice*, which states

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several reasons to debride a chronic wound (**TABLE 12-1**).[13]

CLINICAL CONSIDERATION

The presence of necrotic or devitalized tissue on the surface of a wound prevents accurate assessment of the extent of tissue destruction or depth of the wound.

FIGURE 12-2

Wound with necrotic tissue After a partial forefoot amputation, this foot wound has a large amount of necrotic tissue, both eschar and slough, that requires debridement in order for healing to progress.

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TABLE 12-1

Reasons to Debride Chronic Wounds[12,13]

- Remove necrotic tissue
- Decrease the bioburden
- Remove senescent cells
- Decrease stimulation of inflammatory cell production
- Remove callous, rolled edges (epibole[14])
- Facilitate angiogenesis
- Prepare wound bed for skin equivalents/growth factors
- Prepare wound bed for flap/graft
- Determine depth of tissue destruction

Epibole (**FIGURE 12-3**) is defined as a condition in which the upper edges of the epidermis migrate down (sometimes with a rolled appearance) to envelop the basement membrane or lower edges of the epidermis. Thus, epithelial cells cannot migrate at wound edges and the wound is unable to resurface.[14]

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FIGURE 12-3

Epibole, as seen between the arrows, will impede wound closure and requires debridement of the edges to facilitate epithelial migration.

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During the normal cascade of events leading to wound repair, inflammatory cells such as neutrophils and macrophages are activated to remove microscopic debris, participate in microbial defense, and facilitate the beginning of the repair process. Any interruption in or impairment of the processes associated with the wound repair cascade may create an environment leading to wound chronicity and, ultimately, an environment of increasing necrotic burden.

The term *necrotic burden* refers to the following components:

- Dead or devitalized tissue
- Excess exudate
- Levels of bacteria sufficient to interfere with wound healing.

The necrotic burden is found on the surface of many chronic, non-healing wounds. In addition, resident cells such as fibroblasts and keratinocytes may be phenotypically altered and no longer responsive to certain signals, such as those from chemical mediators, including growth factors. This state of the cell being alive but impaired by its nonresponsive condition is termed *cellular senescence.*[7,15]

TABLE 12-2 lists processes by which necrotic or devitalized tissue impairs and impedes wound closure and healing.

TABLE 12-2

Ways Necrotic or Devitalized Tissue Impairs and Impedes Wound Closure and Healing

- Masking or mimicking signs of infection
- Serving as a source of nutrients for bacterial cells, thereby contributing to the risk of critical colonization or infection
- Acting as a physical barrier to closure and impeding normal matrix formation, angiogenesis, or granulation tissue development
- Impairing contraction of the wound area and epidermal resurfacing
- Stimulating the incessant production of inflammatory cytokines, leading to the overproduction of matrix metalloproteases[2,5,6,7,8–12]

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THE DECISION TO DEBRIDE

There are many clinical and patient-centered factors to consider when making decisions about debridement (**TABLE 12-3**).

TABLE 12-3

Factors for Consideration Before Debridement[2,12,13]

- Overall condition of the patient and their ability to achieve and sustain a closed wound
- Inclusion of the patient's and family's individualized goals for care
- The patient's ability to adhere to the plan of care
- Etiology of the wound
- Type/s of necrotic tissue (ie, slough, eschar)
- Potential of the wound to close and heal due to local factors (eg, blood flow, presence of infection)
- Potential of the wound to close and heal due to systemic factors (eg, nutrition, glucose control, immune status, medications)
- Ability to achieve adequate pain control during debridement
- Clinicians' knowledge, skills, and expertise
- Available resources to support wound care (eg, personnel, reimbursement, availability of equipment and supplies)

The overall condition of the patient and individual goals of care are two primary factors to be considered in the decision to debride.[13] For example, a terminally ill patient with intact, non-infected eschar may not be a candidate for debridement because of comorbidities, medication, and poor cardiac function. The result of debridement may be a larger, potentially more painful wound requiring more extensive topical care with little or no opportunity for closure due to the patient's severely compromised medical status. The patient's general state of health, nutrition, hydration, medications, mobility, and activity status are other factors to be taken into consideration.[13] Patients who take anticoagulants may need a less invasive debridement technique than sharp or surgical debridement. In addition, patients requiring more aggressive techniques are more safely debrided in a controlled setting, such as the operating room or hospital clinic, than at the bedside, in the home, or in the long-term care setting.

CLINICAL CONSIDERATION

Access to adequate anesthesia (topical, regional, or general) must be considered for the patient with intact sensation in the area to be debrided.

Chronic wounds often have underlying pathogenic abnormalities that cause the necrotic burden to re-accumulate. Generally, debridement is not a singular event, but rather combines different methods and/or repeated interventions over time to achieve a clean, functional wound bed. Frequent serial debridements are often necessary to continually stimulate and refresh the surface cells, keeping the wound in a state of readiness to close.[15–17]

The decision to debride requires careful consideration in certain wound etiologies. Stable, non-infected heel or lower-extremity ulcers with impaired perfusion should not be debrided unless signs of infection are present (eg, erythema, fluctuance, separation of eschar from the edge with drainage, or purulence).[13,17] Literature states that stable heel ulcers with black eschar should not be debrided[13]; however, in some instances it may be clinically appropriate to remove stable black eschar on patients who have functional goals and good blood flow. When making the decision to debride stable heel eschar, documentation to support the goals and rationale for performing the procedure are imperative.

CLINICAL CONSIDERATION

- *Vascularity* must be assessed prior to performing any debridement of a lower extremity wound.
- Palpating pedal pulses *only* of patients with lower-extremity wounds is *inadequate* for assessing blood flow.

CASE STUDY

INTRODUCTION

Ms SR is a 62-year-old female resident of a long-term care facility with a past medical history of obsessive compulsive disorder, anxiety disorder,

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hypertension, hyperlipidemia, congestive heart failure, coronary artery disease, chronic obstructive pulmonary disease, and urinary incontinence. She presents to the wound center with an 8-week history of pressure ulcer on the sacral/coccygeal area (**FIGURE 12-4**).

FIGURE 12-4

Sacral pressure ulcer at the time of initial assessment, prior to debridement.

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DISCUSSION QUESTIONS

1. What other information is needed about this patient in order to make a diagnosis?
2. What information is needed to determine the plan of care?

Patients with diabetes who have lower-extremity wounds need more extensive noninvasive vascular assessments (toe pressures [**FIGURE 12-5**] and wave forms, transcutaneous oxygen tension studies, skin perfusion studies, or Duplex arterial studies) to determine blood flow because of the propensity of the more proximal vessels to calcify in people with diabetes.[2,16,19,20] Guidelines for healing potential and for debridement are discussed

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in Chapter 4, Vascular Wounds.

FIGURE 12-5

Toe pressures in patients with diabetes are assessed before debridement.

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Maintaining a dry, stable eschar often leads to slow eschar demarcation and separation as epithelial migration occurs from the edge, eventually closing the wound (**FIGURE 12-6**). This process has been reported in many cases when a patient is not a candidate for debridement. In addition, diagnoses such as pyoderma gangrenosum may result in *pathergy* (defined as enlarging or worsening of the wound), with sharp or surgical debridement (**FIGURE 12-7**).[20]

FIGURE 12-6

Eschar demarcation and separation Dry eschar on a heel with compromised vascular status is sometimes best left in place and not debrided unless there are signs of infection. The epithelium may migrate from the edges under the eschar, which can then be trimmed as it releases. With meticulous care, these wounds will sometimes progress to full closure, albeit quite slowly.

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FIGURE 12-7

Pyoderma gangrenosum Pyoderma gangrenosum has defining violaceous borders around the wound. Wounds suspected of being pyoderma gangrenosum are not sharp or surgically debrided because of the risk of pathergy.

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The clinician who performs debridement must have knowledge of anatomy, understand the patient's overall medical condition, know the wound etiology, and have the technical skills and clinical judgment for debridement, especially when using the more invasive techniques (such as sharp or surgical debridement) in order to have safe, successful outcomes. Clinicians must discriminate viable from nonviable tissue, recognize structures such as arteries and veins, recognize the difference between muscle and granulation tissue, identify fascia covering muscles, identify tendons and cartilage versus slough (which are sometimes similar in color), and identify bone and tendon (see **FIGURES 12-8** to **12-17**).

FIGURE 12-8

Healthy granulation Tissue that is pink/red and moist composed of new blood vessels, connective tissue, fibroblasts, and inflammatory cells that fill a healing wound. Typically, appearing with an irregular, bumpy, or granular surface.

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FIGURE 12-9

Hypergranulation tissue Granulation tissue that is bulbous and friable suggesting heavy bacterial bioburden. Frequently raised above the level of the periwound skin, it may also be seen in wound base, below the periwound surface.

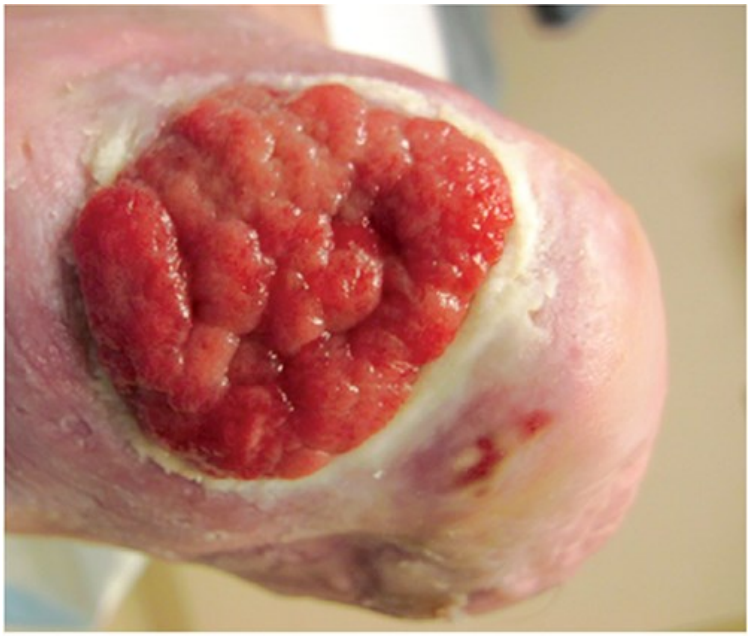

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FIGURE 12-10

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Friable granulation A sign of critical contamination, infection, poor extracellular matrix quality, or overall edema.

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FIGURE 12-11

Slough A metabolic by-product that is composed of serum and matrix proteins; may be white, yellow, tan, brown, or green. It may be loose or firmly adherent and it has a stringy or fibrous texture and appearance has either a soft mushy or stringy fibrous texture, depending on the tissue that has been autolyzed by the phagocytic cells.

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FIGURE 12-12

Eschar Firmly adherent, hard, black, firm, leathery tissue, may be strongly or loosely attached to wound base and edges.

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FIGURE 12-13

Vessel in wound bed Any visible vessels, even if they appear coagulated, are to be avoided in order to prevent bleeding.

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FIGURE 12-14

Muscle/fascia Healthy muscle is bright red and sensate, bleeds when cut, and may twitch when stimulated. Conversely, necrotic muscle does not bleed when cut, is brown and stringy, and does not contract.

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FIGURE 12-15

Subcutaneous fat Fat is poorly vascularized and thus can become necrotic and desiccated easier than other tissue types; therefore, it can become a likely source of bacteria growth.

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FIGURE 12-16

Tendons A tendon that has the sheath with paratenon intact should be protected with a moist dressing to prevent desiccation. Stringy nonviable tendon can be debrided.

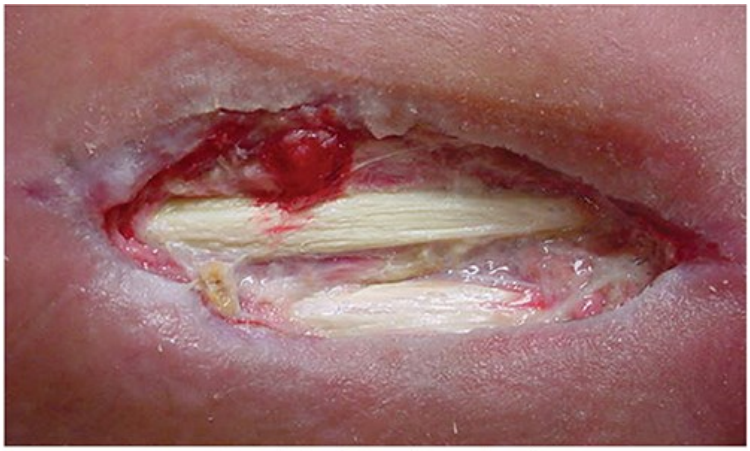

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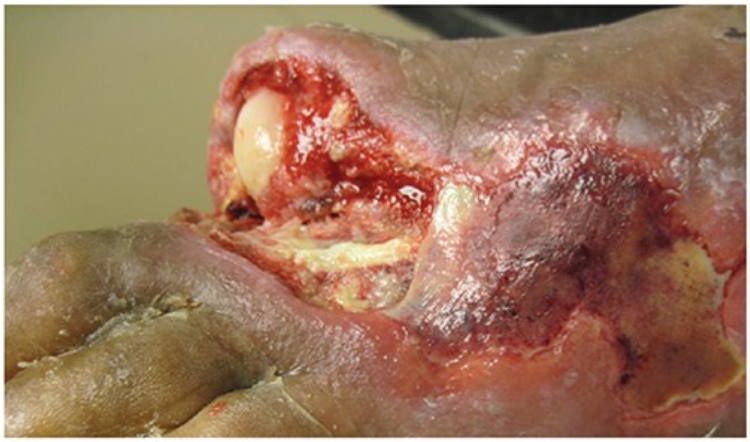
FIGURE 12-17

Bone in wound bed



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In addition to a thorough awareness of the patient's clinical condition, knowledge of the different types of necrotic tissue is necessary to determine the most effective type of debridement to use. Necrotic tissue is generally categorized as slough or eschar; however, any tissue in or adjacent to the wound bed can become necrotic if it loses its blood supply (**FIGURES 12-11** and **12-12**). Therefore, reassessment of the wound is required at every debridement session.

Eschar may be soft or hard, depending on the amount of desiccation (extreme dryness), and is often described as leathery when completely dry. Slough (**FIGURE 12-11**) may be yellow, tan, gray, or brown; is generally moist; and may be loosely or firmly adhered to the wound bed. Slough is a by-product of the autolysis of necrotic tissue and as such, usually has a texture that makes it difficult to grasp with forceps.

The presence of granulation tissue is the hallmark of the proliferative phase of wound repair. Healthy granulation tissue most often appears bright or "beefy" red due to its rich vascular composition (**FIGURE 12-8**). Unhealthy granulation tissue, sometimes called *friable* or fragile granulation tissue, may be a dull, dark red or a pale, pink color.

Hypergranulation tissue (also known as "proud flesh," exuberant granulation tissue, hypertrophic granulation tissue, and hyperplasia of granulation tissue) is a condition that is characterized by an overabundance of granulation tissue, resulting in the extension of this tissue above the level of the wound margins (**FIGURE 12-9**).[18–22] This condition may prevent migration of epithelial cells across the wound surface as these cells do not travel vertically, thereby delaying closure and making the wound more susceptible to infection. In addition, wounds with hypergranulation tissue have an increased risk for excessive scar formation by forcing the wound edges further apart.[22] Malignancies can also present with tissue resembling hypergranulation tissue; therefore, an appropriate referral, biopsy, or both may be indicated.[22] **TABLE 12-4**[2,18,19] provides examples of treatment to diminish or alleviate hypergranulation tissue. **FIGURES 12-18** and **12-19** illustrate the use of polyvinyl alcohol foam to treat hypergranulation tissue.

CLINICAL CONSIDERATION

Malignant tissue that resembles hypergranulation will bleed easily, have a darker red to purple color due to the hypervascularity, and most frequently occurs when there has been a history of hyperplasia.

FIGURE 12-18

Hypergranulation tissue before treatment with PAV foam

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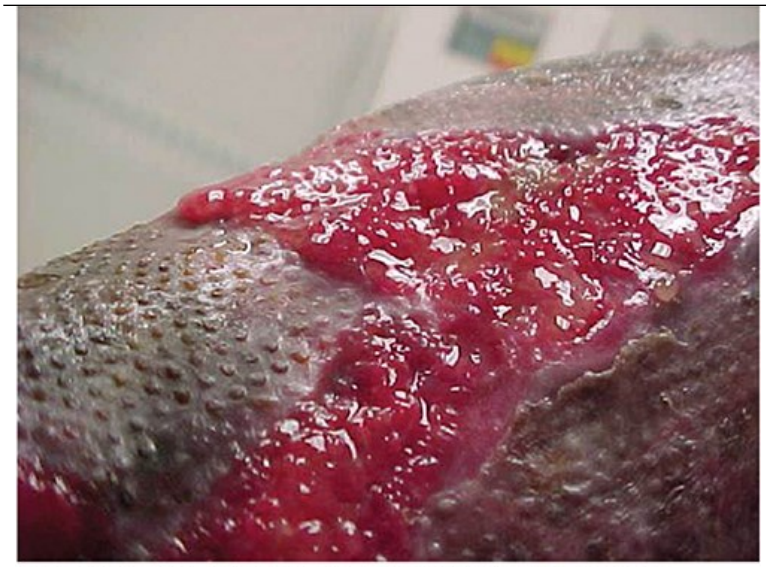

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FIGURE 12-19

Hypergranulation tissue after treatment with PAV foam Note that granulation tissue is more flush or even with wound edges, thereby allowing epithelial migration toward the center of the wound bed.

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TABLE 12-4

Interventions to Reduce Hypergranulation Tissue[2,21,22]

- Compression with dressings such as foams
- Dressings to decrease moisture (eg, hypertonic dressings, calcium alginates, hydrofibers)
- Antimicrobial dressings (eg, silver-impregnated dressings, polyvinyl alcohol foam with organic pigments **FIGURES 12-18** and **12-19** cadexomer iodine)
- Mechanical debridement (eg, scrubbing, low-frequency ultrasound)
- Chemical cauterization with silver nitrate
- Sharp debridement
- Application of topical corticosteroids

Chapter 3 provides more detailed information on tissue types in chronic wounds.

CASE STUDY

A review of the patient's medical history (available through records brought to the clinic with the patient) includes the following:

- Medications: Norvasc, Lipitor, Lasix, two antidepressants, Albuterol
- Vital signs (taken at time of evaluation): BP—100/65; HR—78 bpm; O_2 saturation—94% on room air
- Function: The patient arrives in a wheelchair, records indicate that she is non-ambulatory; transfers from bed to wheelchair with moderate assistance and sits in the chair 3–4 hours each morning and afternoon.
- In addition to regular meals, patient receives protein shakes.
- The patient's wound measurements, using the perpendicular method, are 3.8 cm × 1.7 × 0.1 cm deep.

There is minimal serous drainage on the dressing upon removal and no odor. The patient has visible changes in facial expression and verbal complaints of pain with any tactile sensation to the wound bed.

DISCUSSION QUESTIONS

1. What type of tissue is visible in the wound bed?
2. Describe the edges and the periwound skin.
3. Are any visible signs of infection present?
4. Using the NPUAP Classification for Pressure Ulcer Staging, what stage would be appropriate for this wound?

TYPES OF DEBRIDEMENT

Debridement falls into two general categories: selective and nonselective. With selective debridement, only nonviable tissue is removed; with nonselective techniques, both viable and nonviable tissue may be removed. Debridement is also classified by the method or the mechanism of action of the various techniques that are used to remove nonviable tissue.

The most common types of debridement used in clinical practice include:

- Autolytic
- Enzymatic

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- Mechanical (ie, scrubbing, whirlpool, pulsed lavage, and low-frequency ultrasound)
- Instrument (sharp, surgical, and hydrosurgical)
- Biotherapy or maggot debridement therapy

One or more types of debridement may be used over the course of the wound management to achieve and maintain a clean, functional wound bed.

Autolytic Debridement

Autolytic debridement is the process by which the body's endogenous enzymes (eg, collagenase) loosen and liquefy necrotic tissue in the wound bed (**FIGURES 12-20** and **12-21**). Wound fluid contains these enzymes in addition to inflammatory cells, such as neutrophils and macrophages, that enter the wound site during the normal inflammatory process to remove bacteria and debris.[3,14] Some dressing categories customarily used to provide the optimal environment for autolytic debridement include hydrogels, hydrocolloids, transparent films, foams, alginates, medical-grade honey, and polyacrylate moist dressings (see Chapter 13, Wound Dressings).[2,18–20]

CLINICAL CONSIDERATION

Clinicians working in home health or other settings where dressings are not readily available often ask, “What can I use instead?” For autolytic debridement of the dry eschar, a thin layer of KY jelly can be used instead of hydrogel, under an occlusive dressing to facilitate autolytic debridement.

FIGURE 12-20

Wound before debridement

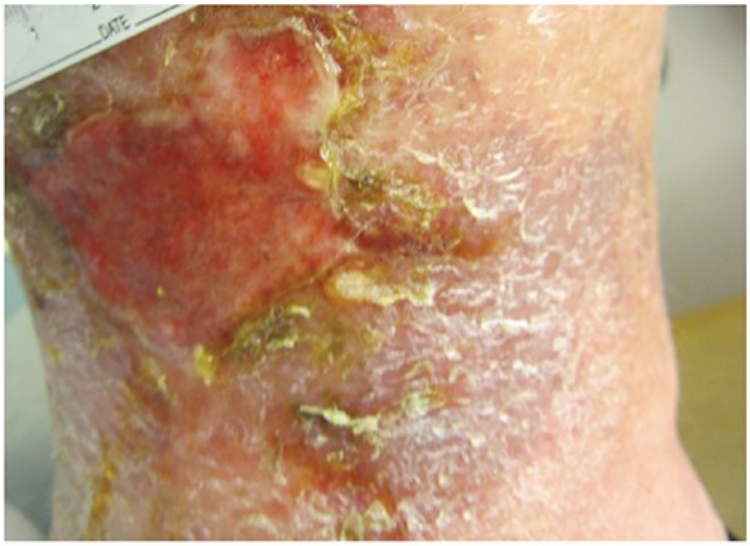

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FIGURE 12-21

Wound after autolytic debridement Note the preservation of periwound skin, flat edges, and lack of necrotic tissue, all a result of the patient's healthy cellular components and enzymes that could break down and phagocytose the nonviable tissue.

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The process of autolytic debridement is slower than other methods, requiring multiple dressing applications and frequently taking weeks to complete. Older or frail patients may have difficulty achieving a clean wound bed using autolytic debridement because of their impaired immune system, aging, comorbidities, and nutrition and hydration deficits. This finding suggests that older patients may need more aggressive forms of debridement than the autolytic process alone.[23]

CLINICAL CONSIDERATION

An adequate immune system and local vascular supply is necessary for autolytic debridement to proceed optimally.

Autolytic debridement is frequently used in combination with other types of debridement, such as mechanical or instrument debridement. While slower, it may be the least painful type of debridement as long as the patient tolerates the dressing changes. Frequency of dressing changes depends on the amount of nonviable tissue to be removed and the amount of fluid that collects under the dressing.

Fluid collection beneath the selected dressing is monitored to prevent prolonged exposure of intact skin, which could lead to moisture-associated skin damage and ultimately create fragile skin that is at risk for further damage (**FIGURE 12-22**). Also, the fluid contains dead cells, cellular debris, and bacteria, frequently causing an odor that may lead to the assumption that the wound is infected, despite the absence of other clinical indicators of infection. Culture of the fluid would likely reveal bacteria that are present but not pathogenic to the wound or patient, possibly leading to unnecessary treatment with antibiotics. The wound surface should be thoroughly and safely irrigated or cleansed after each dressing removal and before assessing for clinical signs of infection.[20]

FIGURE 12-22

Maceration around the wound edges as a result of exposure to the fluids that collect under the dressing used for autolytic debridement.

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Autolytic debridement can be employed to debride wounds of all types, regardless of etiology. However, peripheral arterial disease wounds usually have difficulty with autolytic debridement because of the impaired blood supply and consequent lack of moisture in the lower extremity skin.

Autolytic debridement is frequently the initial intervention in the home and long-term care setting where other, more aggressive forms of debridement may not be feasible or available. The only absolute contraindication to autolytic debridement is the infected wound, which needs to be debrided more urgently.[13] Also, the moisture under the dressing is an ideal environment for bacteria to thrive. Autolytic debridement can be accomplished with minimal technical skills on the part of the caregiver; however, knowledge and skills are required to know if the process is appropriate for a particular wound and patient.

Surfactant Debridement

A unique enhancement to autolytic debridement is the use of a water-soluble Concentrated Surfactant Gel or CSG (PluroGel® Burn and Wound Dressing, Medline Industries, Inc., Northfield, IL). The surfactant, Poloxamer 188, is also often referred to as pluronic F68. Poloxamers are non-ionic synthetic surfactants comprising one central hydrophobic chain and two outer hydrophilic chains. These molecules form loose (non-covalent) cross-links with water to form a micelle matrix (Plurogel micelle matrix or PMM). The matrix is surface active, and in a dynamic system creates a “rinsing” action at a molecular level on the wound bed. This surfactant-driven activity enables the product to disrupt the non-covalent bonds which are instrumental in keeping necrotic tissue and other undesirable elements adherent to the wound bed, thus impairing healing. The product adds moisture more efficiently via the surfactant activity and essentially solubilizes dried exudate and other matter, thereby helping to soften, loosen, and trap wound debris.[24] These undesirable elements can then be rather easily removed at dressing changes via simple rinsing or wiping with wet gauze or similar materials.[25] For thermodynamic reasons specific to the polymer poloxamer, the CSG has a low viscosity appearance when at ambient conditions, particularly in cool ambient conditions (it turns into a liquid in a refrigerator for example), but forms a thick gel as it warms in contact with living tissue. This differentiates it from other common gel like preparations, which are generally viscous when cooled and “runny” when warmed to body temperature. Furthermore, because the surfactant micelles are easily water soluble, the debris-laden gel can be atraumatically washed away during dressing changes.[26]

Lastly, laboratory and clinical studies have demonstrated the ability of the CSG to sequester planktonic microbes and disrupt wound biofilm using the mechanism of action described above.[27,28] The ability of the surfactant to salvage damaged cells has also been extensively reported in literature.[29–31]

Enzymatic Debridement

Enzymatic, or chemical, debridement uses a topical pharmaceutical preparation that is specifically designed to target and break down the devitalized collagen in the wound bed.[14,23,32,33] As with autolysis, enzymatic debridement is an alternative to the more expensive and aggressive methods of

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debridement.

At present there is only one commercially available enzymatic debriding agent in the United States, collagenase, marketed under the trade name Santyl (Smith & Nephew, London, UK).[34] In wound healing, endogenous collagenases belong to a family of extracellular matrix metalloproteases (MMPs) which are naturally occurring enzymes produced by activated inflammatory cells (neutrophils and macrophages) and in certain wound cells (epithelial cells, fibroblasts, and vascular endothelial cells).[8,9,33–36]

Topical collagenase is derived from the bacteria *Clostridium histolyticum.* Reported to be most active in a pH range of 6–8, topical collagenase is specific to native, denatured collagen.[8,15,34] Collagenase promotes debridement by selectively cleaving devitalized collagen strands anchoring necrotic cellular debris without harming viable collagen.[8,15,34] Collagenase has no known effect on viable tissue and is not associated with increased patient discomfort.[15,34]

CLINICAL CONSIDERATION

A 2-mm layer of the collagenase ointment is applied directly to the tissue and covered with an appropriate dressing. The collagenase enzyme is activated by moisture, so the application of a secondary dressing that maintains a moisture balance will allow the enzyme to be released from the ointment.

Collagenase is best applied daily (**FIGURES 12-23** and **12-24**). Cross-hatching of dry or dense eschar is recommended to facilitate penetration of the ointment into subcutaneous tissue and to hasten the debridement process (**FIGURE 12-25**).[20,33] Protection of the surrounding skin with a moisture barrier can prevent maceration and denudation of the intact skin from the increased exudate that results from the enzymatic debridement process. The ointment should not be used with products that could inactivate collagenase (such as detergents or products containing iodine or heavy metals such as lead, silver, or mercury).[34,36] If a wound infection is suspected, the use of an antimicrobial agent per manufacturer's recommendations should be considered for use on the wound surface prior to application of the collagenase ointment.[34]

FIGURE 12-23

Enzymatic debridement Collagenase, available in a thick ointment that is most effective when applied daily, is an enzyme that breaks down denatured protein, thereby releasing the necrotic tissue from the underlying healthy tissue.

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FIGURE 12-24

Application of collagenase[35] The ointment is applied in a thin layer with a sterile applicator and secured with a moist occlusive dressing that will

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both provide moisture to activate the enzymes and contain the resulting exudates and debris.

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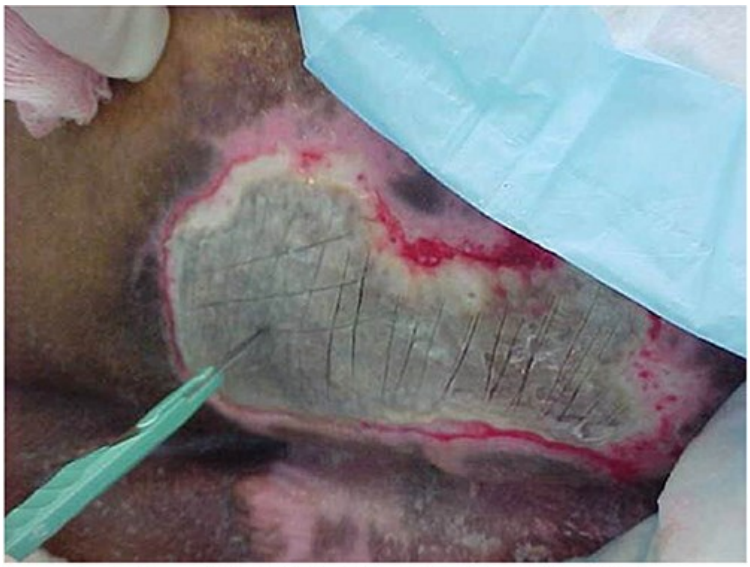
FIGURE 12-25

Cross-hatching of eschar Both autolytic and enzymatic debridement of eschar is facilitated by cross-hatching the surface of the necrotic tissue with a scalpel prior to application of the dressings.



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While initial debridement is important to remove necrotic tissue as a barrier to healing, maintenance or ongoing debridement may be needed to prevent the re-accumulation of debris. Apoptosis, or programmed cell death, is a recurring process in wounds.[15] Continued use of collagenase may address this re-accumulation, thereby allowing the proliferation of healthier granulation tissue.[15,23]

Choosing between autolytic and enzymatic debridement is based on the following:

- Patient condition
- Speed at which debridement is desired

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- Access to products
- Type of tissue to be debrided
- Ability of the patient's body to support the autolytic debridement process

Autolytic debridement, while effective in some patients, is a more passive approach than enzymatic debridement. Milne et al.[37] conducted a randomized controlled trial of 27 patients with pressure ulcers with nonviable tissue, who had received no previous debridement or cross-hatching. The collagenase group was found to have statistical significance in achieving full debridement by day 42. Additionally, the collagenase group had a greater reduction in nonviable tissue on a weekly basis as compared to a control group receiving autolytic debridement with hyrogel.[37]

Mechanical Debridement

Mechanical debridement is the use of external forces or energy directly to the wound surface to dislodge and remove debris, bacteria, and necrotic burden. Depending on the method used, this type of debridement can be selective (nonviable tissue only) or nonselective (viable tissue included in process). The most common types of mechanical debridement include

- Soft abrasive debridement
- Various forms of hydrotherapy
- Wet-to-dry or wet-to-moist dressings
- Low-frequency contact ultrasound

Soft Abrasion Debridement

Soft abrasion debridement uses dry gauze or a cotton- or alginate-tipped applicator to gently lift and remove non-adherent debris and congealed surface exudates.[2] Pain that sometimes occurs may be mitigated with use of a topical anesthetic, which needs to be applied at least 15 minutes prior to treatment for maximum benefit. Abrading the wound edge of new advancing epithelial cells may be used to prevent the formation of epibole.[2] Other indications for soft abrasion are scales that result from chronic venous insufficiency, debris after autolytic or enzymatic debridement, coagulum in the wound bed, or any other loose devitalized tissue.

CLINICAL CONSIDERATION

A fibrous debridement pad (Debrisoft by Lohmann-Rauscher, Rengsdorf, Germany) is available in a 10 × 10 cm pad or a smaller pad on a lollipop stick, and is excellent for debridement of wounds in the pediatric population.

Hydrotherapy

Hydrotherapy for debridement purposes primarily includes whirlpool and pulsatile lavage with suction.

Whirlpool

Whirlpool (WP) is considered a nonspecific, *nonselective* form of mechanical debridement that facilitates cleansing by immersing the patient's body part in a tub or tank while a turbine motor agitates the water to loosen the necrotic tissue (**FIGURE 12-26**). *WP treatment is no longer a recommended mechanical debridement treatment for wound care* (**TABLE 12-5**).[38] The risks of infection from cross-contamination and aerosolization are hazards that must be considered.[5] Biofilm growth is known to occur not only in wounds but also in medical equipment such as WPs.[38,39]

FIGURE 12-26

Whirlpool The agitators in the whirlpool produce mechanical forces in the water that can help soften nonviable tissue and cleanse a wound. However, the risks for skin and tissue harm usually are greater than the benefits.

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TABLE 12-5

Contraindications to Whirlpool Therapy for Debridement or Wound Cleansing[2,38–40]

- Potential for biofilm growth in WP and associated plumbing
- Potential cross-contamination by bacteria (eg, *Pseudomonas aeruginosa*)
- Inability to determine if WP pressure delivered to wound surface is damaging to granulation tissue
- Maceration of tissues from water (particularly detrimental to diabetic feet)
- Increased venous hypertension in presence of venous insufficiency

In most cases, WP is an inappropriate intervention for chronic venous insufficiency and diabetic foot ulcers.[2,19,33,38,39] Chronic venous insufficiency wounds tend to be wet due to their highly exudative nature. WP creates the potential to exacerbate the often-macerated condition of the periwound tissue. McCulloch and Boyd[39] demonstrated that the lower extremities of patients with chronic venous insufficiency ulcers immersed in the WP for longer than 5 minutes with the leg in the customary dependent position resulted in increased venous hypertension and vascular congestion leading to an increase in limb edema.

For patients with diabetes who have peripheral neuropathy, the prolonged exposure of the foot to warm water during WP causes skin maceration, leaving the foot more susceptible to injury.[2] In addition, the warm water contributes to the anhidrotic, dry skin condition frequently present in this patient population by washing away the already-diminished body oils that assist in protecting the skin. The current standard care is to *not* soak the diabetic foot, and WP is a “soaking” intervention.[40]

Pulsatile Lavage with Suction

Pulsatile lavage with suction (PLWS) incorporates the energy of a pulsating mechanical force delivering saline (or other desired solutions) to loosen and cleanse the wound bed, with simultaneous suction for removal of the saline irrigant, loosened debris, microorganisms, and exudate (**FIGURE 12-27A** and **TABLE 12-6**).[2,14,33] PLWS has eliminated the use of WP in most practice settings. This biophysical energy is highly effective in dislodging slough and can be a selective form of debriding necrotic tissue when the lavage force is targeting nonviable tissue only.

CLINICAL CONSIDERATION

Whirlpool is an inappropriate intervention for patients with chronic venous insufficiency and diabetic foot ulcers.[2,19,33,38–41]

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FIGURE 12-27

A. Pulsed lavage with suction A flexible tip allows the clinician to mechanically debride a variety of wound sizes and shapes with PLWS. The clinician uses the fingers to control the contact of the tip with the wound surface, thereby avoiding too much pressure on the tissue as well as preventing unnecessary splashing of contaminated water. The technique requires practice and knowledge of wound healing in order to obtain optimal outcomes. (Used with permission from Harriett Loehne, PT, DPT, CWS, PLWS with splash shield.) **B. PLWS protective equipment** A plastic sleeve to cover the reusable hand-piece and a plastic sheet to cover the area being treated are two equipment adaptations that help prevent unnecessary splashing and aerosolization of contaminated water. (Davol, a Bard® Company. Davol is a registered trademark of C.R. Bard, Inc. Used with permission.)

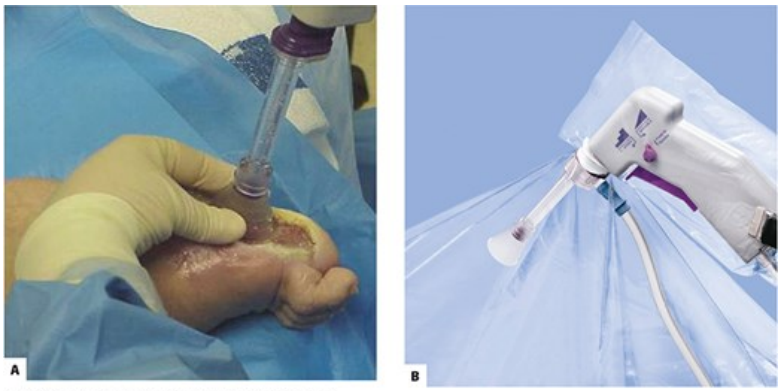

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TABLE 12-6

Proposed Benefits of Pulsatile Lavage with Suction

- Provides controlled pressure below 15 psi guidelines for optimal cleansing and irrigation.
- Efficient removal of necrotic tissue, bacteria, and foreign material.
- Site-specific treatment avoids possible maceration of adjacent tissue or viable tissue.
- Portable. Can take PLWS units to any care setting.
- Easy setup.
- Single patient use—eliminates sterilization and equipment maintenance.
- Minimizes potential for cross-contamination or staff injuries resulting from lifting and moving patients.
- Convenient premade kits available.
- Splash sheets available minimize aerosolization of wound fluids, thereby protecting clinician, patients, and environment.
- Various application tips for use in different types of wound beds (ie, tunnels, tracts, fistulas).

This method of debridement is one of the few that can function in tracts, tunnels, and areas of extensive undermining using specialized tips.[33] Pressure settings are below 15 pounds per square inch (psi) to prevent driving the bacteria into underlying soft tissue and to prevent damage to granulation tissue present in the wound bed.[2,13,33] See Chapter 17, Pulsed Lavage with Suction, for specific details on techniques, indications, and contraindications.

Personal protective equipment is necessary for the clinician performing PLWS. In addition, treatment should be delivered in an enclosed, private treatment area to avoid contamination by aerosolization.[2,33,41] A flexible polyurethane protective shield to cover the wound area and instrumentation can be used to assist in minimizing the aerosolization associated with PLWS (**FIGURE 12-27B**). Research has not been performed on this protective device to demonstrate if aerosolization is completely contained. The clinician must be experienced using PLWS when treating complex wounds, such as those with fistulas, exposed cavity linings, and long tunnels into body cavities.[2,33]

When performing PLWS on wounds with tunnels, the clinician closely monitors the amount of fluid delivered to and suctioned from the wound to ensure that fluid is not going into a body cavity.

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Wet-to-Dry Dressings

Wet-to-dry dressings are a nonselective method for debridement of necrotic tissue. This dressing technique, normally referred to as either wet-to-dry or wet-to-moist, unfortunately remains one of the most common forms of debridement in many care settings.[42–44] The name is somewhat misleading in that the gauze dressing is rarely placed into the wound bed in what would be considered a wet level of hydration. Rather, the most commonly discussed procedure is to wet open-weave gauze, squeeze or wring out the fluid until it is just moist, and then open and place the gauze into the wound bed such that a layer of the gauze is in intimate contact with all areas of the wound surface. The proposed mechanism of action is that as the gauze dries, it becomes adherent to the wound surface, and the surface debris are torn from the wound bed as the dressing is removed (**FIGURE 12-28**).[2,44]

FIGURE 12-28

Wet-to-dry debridement Removal of a wet-to-dry dressing over healthy granulation tissue disrupts fragile capillaries and damages new granulation tissue.

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This nonselective form of debridement, which can damage healthy granulation tissue and structures in the wound bed, continues to be a controversial issue in wound management. The technique has many disadvantages that may overshadow any potential benefit. These disadvantages include, but are not limited to, the following:

- The procedure is nonselective as a form of debridement and indiscriminately removes any tissue with which it is in contact.
- Gauze does not have to completely dry to cause trauma to the wound surface. The very absorption of exudate from the wound surface causes fibers to become embedded in the wound bed and consequently to adhere upon removal of the gauze.
- Wet-to-dry dressings often cause pain upon removal.

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- Gauze as a packing material is often over-packed, causing increased detrimental pressure on the surface of the wound, thereby increasing the likelihood of further tissue necrosis.
- Exposed structures such as bone, muscle, or tendon are likely to desiccate using a wet-to-dry procedure.[2,13,42–44]

Although the materials for wet-to-dry debridement are inexpensive and readily available, this procedure can actually be more expensive than current advanced dressing choices and debridement techniques because of the increased clinician time required for each dressing change and the more frequent dressing changes required, as well as prolonged time to heal.[43]

CLINICAL CONSIDERATION

Wet-to-dry dressings are absolutely inappropriate for wounds with significant amounts of healthy granulation tissue in the wound bed.

Additionally, wet-to-dry dressings may impede wound healing due to local tissue cooling; disruption of granulation tissue by dressing removal; and increased infection risk from frequent dressing changes, strike-through drainage, and prolonged inflammation.[43–45] One study found that wet-to-dry dressings were inappropriately prescribed for wounds not needing debridement in more than 78% of the wounds for which they were ordered.[44]

All of the negatives aside, when performed properly, wet-to-dry debridement can be a relatively quick and often effective method for removing devitalized tissue from a wound bed that is predominately covered with necrotic tissue and has adequate blood flow. Caution is advised with anticoagulated patients as bleeding may occur with dressing removal. As with any wound dressing change, adequate pain control is necessary for successful treatment.[2,13] For massively large wounds, moistened gauze is often the only packing material that can reasonably and cost-effectively fill a large defect. However, once granulation tissue begins to form in the wound bed, the clinician should shift from the wet-to-dry technique to another intervention (eg, negative pressure wound therapy) that will not damage the new, fragile tissue and will facilitate faster closure. If negative pressure wound therapy is not available, a thin layer of hydrogel over the wound surface before filling with a moist gauze dressing will help prevent adherence to the viable tissue and decrease pain with removal.

Low-Frequency Ultrasound Energy in Debridement

In recent years, low-frequency ultrasound (LFU) energy has been employed to positively impact tissues in the chronic wound by performing debridement and facilitating wound closure and healing.[13,45] There are several devices, manufactured by various companies, which provide different methods of delivering sonar energy to the wound for bactericidal effects, and with the *contact* devices, immediate selective debridement of necrotic tissue is achievable. In addition, *non-contact* forms of low frequency ultrasound are thought to facilitate wound closure and healing processes.[45–48] (Please see Chapter 16, Ultrasound, for a discussion of ultrasound energy in wound care.)

Use of low-frequency *contact* ultrasound for wound debridement has several advantages:

- Immediate results when using contact LFU
- Pain management with only topical anesthesia
- Selective debridement of necrotic tissue; nonselective debridement of *unhealthy* viable tissue, and undesirable tissues such as rolled or thickened non-migrating edges (**FIGURES 12-29** and **12-30**)

As mentioned earlier, LFU is bactericidal at the surface and penetrates several millimeters into deeper tissues directly beneath the probe tip. It can be performed in a variety of settings by trained personnel, permitting energy delivery at the bedside, in the outpatient setting, or during operative procedures. In addition to separating dead tissue from the wound bed, contact LFU has the following positive effects that contribute to wound closure and healing:

- Increased local tissue perfusion via vasodilation and resolution of vasospasm (thermal effect)
- Decreased bacterial load
- Stimulation of fibroblasts, macrophages, and endothelial cells to promote healing[45–51]

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FIGURE 12-29

Ankle wound before contact LFU debridement

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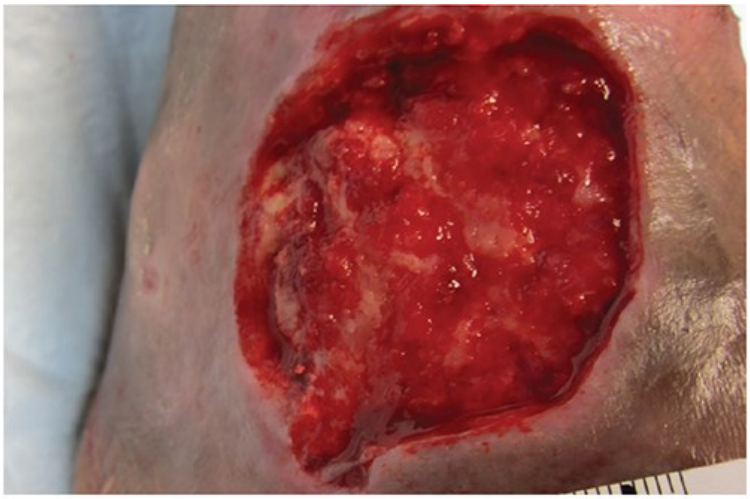
FIGURE 12-30

Wound after contact LFU debridement Note wound edges are debrided to fresh tissue to facilitate keratinocyte migration for wound re-epithelialization.



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Sharp/Surgical Debridement

Sharp/surgical debridement refers to the use of instruments or devices capable of excising or cutting away necrotic tissue and surface debris. The instruments include, but are not limited to, forceps, scalpels, curettes, and scissors (**FIGURES 12-31** to **12-34**). The instruments used for sharp/surgical debridement may be reusable or disposable; however, those found in the average suture removal kit are generally not strong or sharp enough to adequately accomplish wound debridement. (An exception is the suture removal kit that contains scissors that are pointed and sharp enough to be termed iris scissors.) In addition, surgical debridement may be accomplished using a device such as the high-powered parallel waterjet.

CLINICAL CONSIDERATION

Prior to mechanical or sharp/surgical debridement, a review of the medications for anticoagulant therapy is imperative. Excessive, potentially dangerous bleeding can occur with patients on blood thinners or with low platelet counts, thereby creating an emergency situation.

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FIGURE 12-31

Scalpels Each numbered blade has a different shape that makes it useful for different types of tissue and wound shapes.

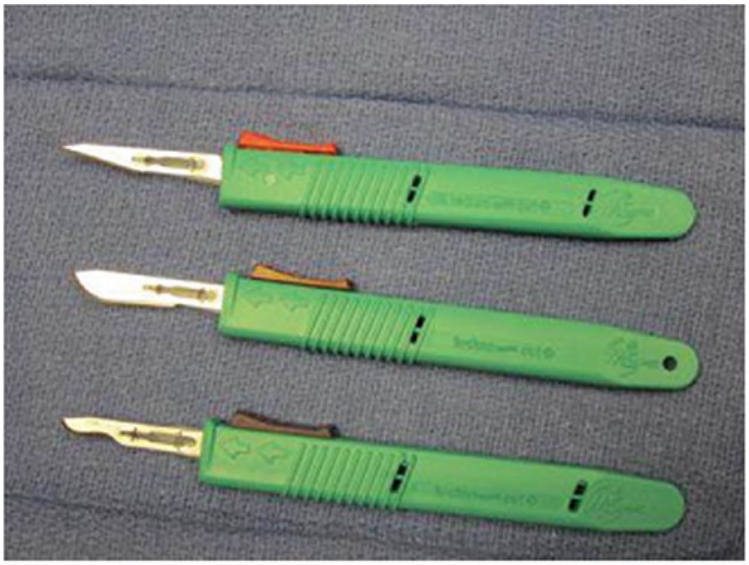

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FIGURE 12-32

Example of serrated (A) and standard forceps (B)

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FIGURE 12-33

Examples of curved (A) and straight iris (B) scissors The small pointed tip allows for precise cutting of necrotic tissue in small areas.

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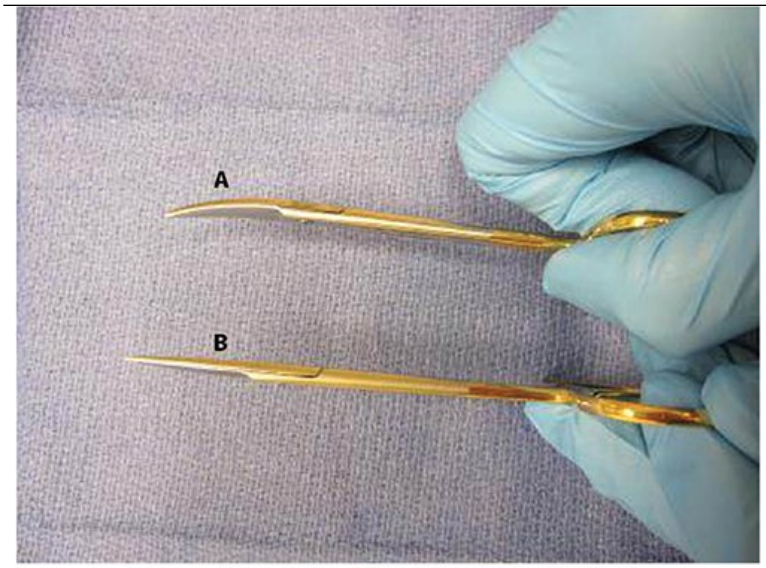

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FIGURE 12-34

Curretts Examples of disposable curretts useful for sharp debridement of slough and devitalized adipose tissue.

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Sharp/surgical instrument debridement (**FIGURE 12-35**) is a rapid method of removing necrotic tissue. Conservative sharp debridement may also be performed in serial sessions, potentially combined with another form of debridement, with the goal of creating a functional clean wound bed. Callous debridement on diabetic feet is mandatory to prepare the wound bed for closure and is performed with a scalpel using a shaving motion and with forceps and scissors to lift and cut (**FIGURE 12-36**).

CLINICAL CONSIDERATION

Knowledge of one's scope of practice as defined by the state practice act is paramount before performing sharp debridement. In general, nurses and physical therapists perform sequential or serial conservative sharp instrument debridement, ensuring that only nonviable tissue is removed, whereas physicians, podiatrists, nurse practitioners, and physician assistants may perform both conservative sharp and surgical/excisional debridement that may or may not extend into unhealthy viable tissue.[20]

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FIGURE 12-35

Sharp debridement of pressure ulcer eschar Performing sharp debridement of a pressure ulcer using the lift and cut technique. The forceps are used to lift the edge of the eschar so the scalpel or scissor cut can be made precisely between nonviable and viable tissue.

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FIGURE 12-36

Callus debridement Removal of callus from a plantar metatarsal head. Note the wound under the debrided callus.

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The clinician performing instrument debridement should have adequate training and skill competency validation. Physicians, especially surgeons and podiatrists, are well prepared as a result of their education and training. Other providers (nurses, physical therapists, physician assistants) require additional didactic and skills lab training followed by additional hands-on preceptorship before performing routine sharp debridement. Even if the training is part of the educational curriculum, validation of skills and competencies according to facility procedure is required, and evidence of completion of such training should be maintained in the clinician's employee file.[20]

The patient must be adequately prepared for sharp/surgical debridement. Informed consent is required for surgical debridement (often referred to as

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incision and drainage or I&D). The facility or agency protocol dictates consent policies, and the clinician should carefully adhere to these policies. Prior to sharp/surgical debridement, the wound site is prepared by adequate cleansing to remove exudate, residue, and any loose debris that may have accumulated since the previous dressing change. This is a necessary step to avoid contamination of the wound bed with the flora present on periwound skin. Chlorhexidine wipes are also useful for cleansing the periwound skin prior to debridement.

Adequate pain management both during and after the procedure is imperative for patient-centered care.[12,20,52] Although devitalized tissue is devoid of sensation, the actual procedure may induce discomfort of various degrees. Preprocedure instructions include advising the patient to inform the practitioner when they feel discomfort, thus giving the patient a sense of control during the debridement process. Debridement is stopped as soon as the patient expresses unacceptable discomfort. This level varies greatly among patients, and strategies for pain management are individualized. Premedication can help reduce the patient's anxiety and minimize discomfort related to cleansing, preparing the wound for debridement, and performing the debridement procedure. Oral or intravenous medications can be prescribed by the physician and administered by the nurse in an inpatient setting. Additionally, topical or local anesthetics (**FIGURE 12-37**) may be employed after adequate cleansing to reduce or eliminate procedural pain in sensate tissues. Finally, there should be a plan for adequate pain management once the procedure is completed.

FIGURE 12-37

Examples of analgesics Topical analgesics are used to decrease pain during debridement of sensitive wounds. A thin layer is applied to the wound bed and edges and allowed to sit for 10–15 minutes before beginning the procedure. Two percent Lidocaine sprays are now available without a prescription.

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The decision to perform surgical debridement in the operating room is based largely on the extent and depth of the required debridement and the need to accomplish the procedure under controlled conditions (eg, due to pain, bleeding, sepsis). Other factors may include the following:

- The need for emergent procedures such as in the case of infected wounds or patient sepsis
- The need for a higher degree of asepsis, such as the potential exposure of tendon, bone, or joints
- The need for extensive bone debridement
- The need for adequate anesthesia
- The presence of extensive undermining, sinus tracts, or tunneling
- The potential for excessive bleeding

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- The patient's anxiety or stress related to the procedure[20]

In addition to the use of instruments, the operating room provides an environment for the use of devices that accomplish debridement into deeper tissues more rapidly than with instruments, including the use of the high-powered waterjet.

Hydrosurgical Debridement

The high-powered parallel waterjet (Versajet, Smith Nephew, Largo, FL) is a more recent device for performing surgical debridement. This tool precisely removes nonviable tissue using a high-energy water stream. It is used only by a skilled surgeon in an operating room environment (**FIGURES 12-38** and **12-39**). The waterjet is a Food and Drug Administration–approved medical device that has the ability to focus a high-powered stream of water into a high-energy cutting implement. The saline used in the waterjet is enclosed in a sterile circuit that passes through a small but powerful pump. This cutting action provides the surgeon precise control over the wound surface.[53,54]

FIGURE 12-38

Hydrosurgical debridement Wound with a large amount of slough is appropriate to be debrided with Versajet. (Used with permission from Greg Patterson, MD, FACS, CWS, FASA.)

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FIGURE 12-39

Clean, healthy wound bed after debridement with Versajet (Used with permission from Greg Patterson, MD, FACS, CWS, FASA.)

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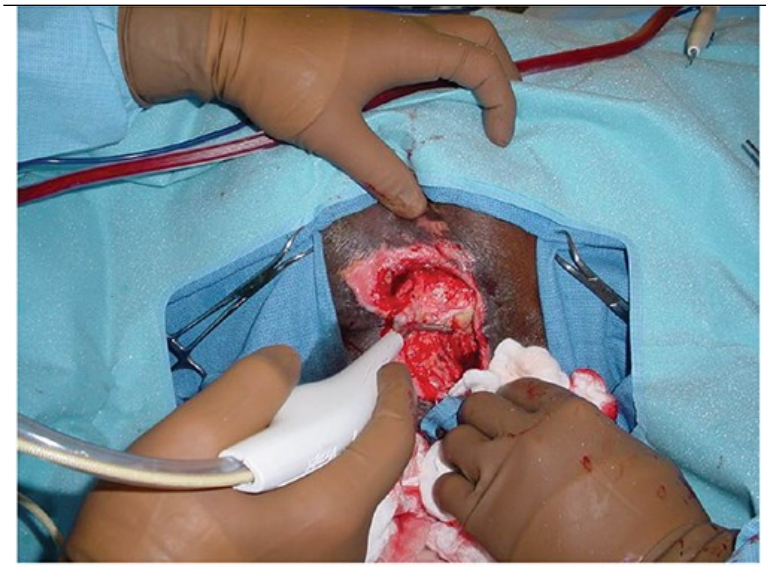

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This form of surgical debridement is reported to be less effective in pressure ulcers that are covered with dry eschar.[53] The preferred strategy is to remove the eschar by sharp debridement and follow with the use of waterjet to debride the underlying necrotic tissue. Consequently, all of the necrotic tissue, fibrinous debris, and unhealthy tissue can be removed with no injury to the healthy underlying collateral tissue.[53] Surgeons can perform more aggressive wound debridement while simultaneously removing less surrounding healthy tissue.[53,54]

Bleeding in Mechanical and Sharp/Surgical Debridement

Bleeding is always a possibility as a result of debridement by mechanical means or with the use of instruments. Small amounts of punctate bleeding are expected and easily controlled with either time or gentle pressure. If bleeding continues, the site is elevated and firm pressure applied to the wound with gauze or a gloved finger for 5–10 minutes *without lifting the gauze to look because this may dislodge the clot and cause more bleeding to occur*. If there is one small site of bleeding, using a sterile applicator to apply direct pressure at the specific location may be effective. If bleeding continues, hemostatic agents may be applied, including calcium alginates, silver nitrate, dressings containing chitosan, or thrombin agents such as Gelfoam (Pfizer, Inc, New York, NY), Surgicel (Ethicon, Inc, Somerville, NJ), or QuikClot (Z-Medica Corporation, Wallingford, CT). If bleeding does not stop with the methods above, the physician should be notified for further intervention such as sutures or electrocautery.[2,20] If the bleeding is pulsatile, indicating that it is coming from an artery, the physician should be called immediately as only suturing will halt arterial bleeding. Pressure is maintained while waiting for the sutures to be applied.

CLINICAL CONSIDERATION

Bleeding is always a concern when performing sharp or surgical debridement and is managed by having a hemostatic agent readily available during the procedure.

Checking the patient's medication list prior to debriding is imperative. Often people needing debridement are on anticoagulant therapy, which causes longer clotting times, thereby creating potential for excessive bleeding with sharp/surgical or mechanical debridement. Low platelets can also cause excessive bleeding, therefore checking lab values is an integral part of the pre-debridement assessment especially in the acute care setting. Conservative types of debridement are preferred for these patients. If large amounts of tissue need to be removed, surgical debridement by a physician in the operating room is advised.

Maggot Debridement Therapy

Maggot debridement therapy (MDT) (also known as larval therapy, biotherapy, biosurgery, and biodebridement) is the purposeful use of maggots in wound care (**FIGURE 12-40**).[55,56]

FIGURE 12-40

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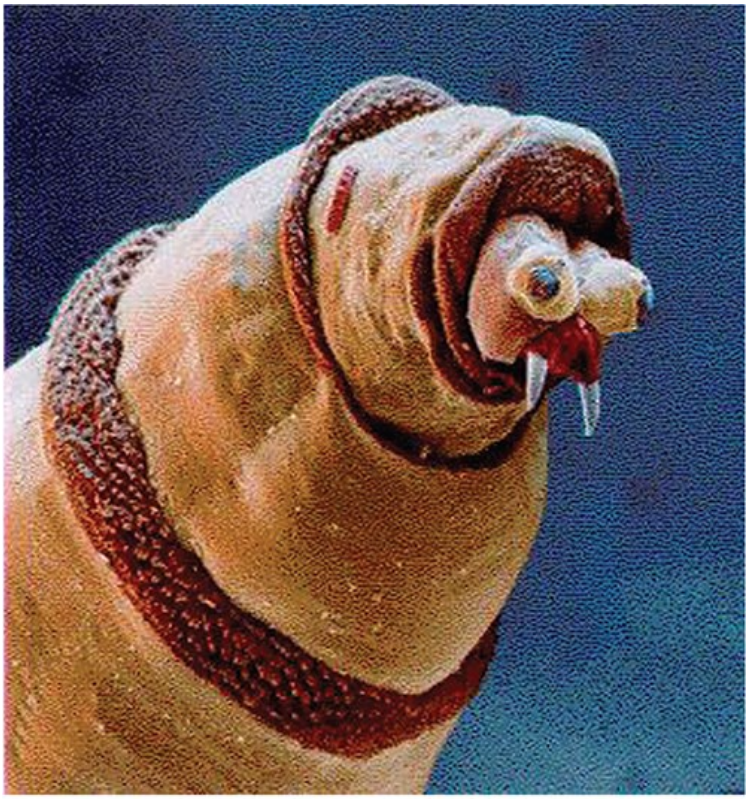
Maggot used for debridement Photo of maggot under color scanning electron micrograph (SEM). Maggots feed on necrotic tissue only creating a debridement effect and have antibacterial chemicals in their saliva and excrement to help fight infection.



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The benefits of maggot therapy have been documented as far back as the 16th century.[2,55] Military surgeons noted that soldiers whose wounds became infested with maggots had a much lower mortality rate and exhibited cleaner, faster-healing wounds than did soldiers with similar wounds that were not infested. During the late 1920s, William Baer, an orthopedic surgeon at Johns Hopkins University, began to systematically treat, study, and publish case studies of patients using MDT.[55] Weil et al.[56] suggested using the term *larval therapy* in 1933, and up to 1936 there was a massive growth in popularity. With the introduction of penicillin in the 1940s, larval therapy was relegated to the archives of "old-fashioned" treatments.[55] In the 1980s, Sherman[57,58] began to reintroduce larval therapy following his research in the treatment of pressure ulcers and later Dumville et al.[59] reported use with chronic venous ulcers on the lower extremity. The rationale, application, and benefits reported were no different than those of Baer 64 years earlier. The positive results sparked a new wave of interest in this historical treatment, and a surge in research followed. By the mid-1990s, there were reports of approximately 60 centers in the United Kingdom using maggots to treat intractable wounds. Larvae were being used to treat chronic necrotic wounds with significant slough that were not responsive to conventional treatment such as hydrogel dressings and surgical debridement.[60] A resurgence of maggot therapy has occurred in recent years, as more widespread acceptance among the medical community, as well as patients, has occurred.

Maggots have three proposed actions: debridement of necrotic tissue, antimicrobial activity, and facilitation of wound healing. They are indicated for debriding non-healing necrotic skin and soft tissue wounds including pressure ulcers, chronic venous insufficiency ulcers, neuropathic foot ulcers, and non-healing traumatic or post-surgical wounds.[57–64] Maggots selectively debride by feeding on the necrotic tissue, cellular debris, and exudate in wounds. The maggot secretes trypsin-like collagenases and chymotrypsin-like enzymes, which lyse the necrotic tissue into a semi-liquid form that the creatures can ingest.[65]

CLINICAL CONSIDERATION

Maggots have been used successfully to debride

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- Pressure ulcers[57,58]
- Venous insufficiency ulcers[59]
- Diabetic foot ulcers[60,61]
- Infections including necrotizing soft tissue infections[62–64]

To attach to tissue and provide locomotion, maggots use a pair of mandibles or hooks. The maggot also uses these hooks during feeding to disrupt membranes and thus facilitate the penetration of its proteolytic enzymes.[55] Additional debridement may be facilitated by the maggots crawling about within the wound, dislodging small amounts of necrotic material.

The ability of maggots to kill or prevent the growth of potentially pathogenic bacteria has been the subject of a number of studies. Marked antimicrobial activity has been detected against *Streptococcus* A and B and *Staphylococcus aureus*, with some activity detected against *Pseudomonas* and a clinical isolate of a resistant strain of *S. aureus* (MRSA).[62,64]

Growth of granulation tissue has been reported to be faster with better wound healing rates using maggot therapy. One study suggests that a possible mode of action for facilitating wound healing is the increase in tissue oxygenation that takes place when using MDT.[66]

In 2004, the United States Food and Drug Administration approved the production and marketing of medical grade maggots in the United States as a medical device. The technique for application and use of MDT is not difficult but is specific; there are two methods of delivery of the larvae to the wound bed. In the original method, the wound bed is cleansed and prepared, and the surrounding skin is dried thoroughly and protected with a skin barrier wipe. A combination dressing supplied by the manufacturer to secure and trap the larvae may be used or the clinician may create an appropriate dressing. The wound is "picture framed" by building one or two layers of hydrocolloid or pectin skin barrier strips along the wound edges. Commonly, practitioners also apply skin contact cement to the edges. The maggots are applied to the wound with five to eight maggots per cm^2 of wound surface area. A closed-mesh or veil dressing is applied and adhered to the cement-edged barrier, thus preventing migration of the maggots outside of the wound area. The wound is further dressed with gauze or other absorbent materials to adequately manage the increased exudate that generally accumulates. The dressing is left in place for 48–72 hours and the maggots are removed as they become satiated and move to the surface of the dressing. A newer delivery system utilizes a "bagged" system where the larvae are delivered to the wound bed in a sealed bag sized appropriately to the wound, and if needed using more than one bag. The activity and mechanism of action of the maggots is unchanged; however, the ease of use is enhanced by one-step application and removal. One or two cycles of MDT are applied each week. The total duration of treatment depends upon the size and character of the wound, the clinical response, and the goals of therapy. Instrument debridement of thick, dry eschar or tough, fibrous tissue prior to initiation of MDT hastens the effect and shortens the treatment time (**FIGURES 12-41** to **12-46**).

CASE STUDY

CONCLUSION

A topical anesthetic gel was applied for pain management, and debridement was attempted with a curette. The patient was unable to tolerate the instrument debridement due to pain assessed as severe. Contact LFU was initiated and 50% of the nonviable tissue was removed. The ulcer was then assessed as a Stage III (**FIGURE 12-47**). The patient began to get very anxious and agitated, both indications to terminate this treatment session. Collagenase ointment was ordered for the nursing staff to apply daily and cover with a foam dressing. The patient returned to the long-term care facility with an appointment to return in 1 week. Because of the patient's anxiety disorder, the goal was to get the wound debrided as quickly as possible and reduce the frequency of visits to the wound center.

FIGURE 12-47

Case study wound on the first visit after debridement with contact, low-frequency ultrasound

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Goals:

1. Wound will be debrided of all necrotic tissue and progress to the proliferative phase of wound healing.
2. Patient pain levels will be less than 4 on the Faces Pain Scale.
3. The nursing staff will be consistent in repositioning patient in both supine and sitting positions.

Second visit (after 1 week of treatment)

At the time of the second visit 1 week later, the wound measured 3.1 cm × 1.6 × 0.1 cm. Scattered slough and fibrinous debris remained on the wound surface. A topical anesthetic was applied for 15 minutes, a curette was used to release the edges of the wound, which were beginning to roll with epibole; however, the patient complained of pain and became agitated, thus the debridement process was stopped. Orders were written for the nursing staff to continue the collagenase ointment, and an appointment was made for 1 week later.

DISCUSSION QUESTIONS

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1. Describe the tissue, edges, and periwound skin in **FIGURE 12-48**.
2. What is the wound healing phase?
3. What is the wound stage (based on the information provided in Chapter 6 on Pressure Injuries in the healing process)?

Third visit (after 2 weeks of treatment)

FIGURE 12-48

Presentation of the wound at the time of the second visit, after removal of the dressing and before treatment was attempted
Debridement was aborted due to patient complaint of pain and increasing agitation.

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The wound is now cleaner as the enzymatic debriding agent continues to lyse and clean the wound bed. Patient tolerates the cleansing and reassessment with no complaints of pain. The wound now measures 2.7 × 1.1 × 0.1 cm. Nursing staff is to continue the same plan of care. Goals are revised to full closure by secondary intention. Nursing staff is to continue repositioning and monitoring of nutritional status to ensure no recurrence.

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DISCUSSION QUESTIONS

1. Describe the tissue, edges, and periwound skin in **FIGURE 12-49**.
2. What phase of wound healing is evident?

FIGURE 12-49

Presentation of the wound after 2 weeks of treatment with enzymatic debridement

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Fourth visit (after 3 weeks of treatment) (FIGURE 12-50)

FIGURE 12-50

At the time of the fourth visit, the wound is covered with new epithelial tissue

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The wound has made excellent progress in the last week presenting with a small open area, mostly covered with newly formed epithelium. Scant drainage is noted on the dressing after removal and the patient expresses no pain during the treatment session. A foam dressing is ordered for protection from friction and incontinence moisture. The patient is scheduled return for a final assessment in 1 month.

Fifth visit (after 4 weeks of treatment) (FIGURE 12-51)

FIGURE 12-51

At the time of the fifth visit, the newly closed wound has fragile epithelial tissue and is in the remodeling phase of healing

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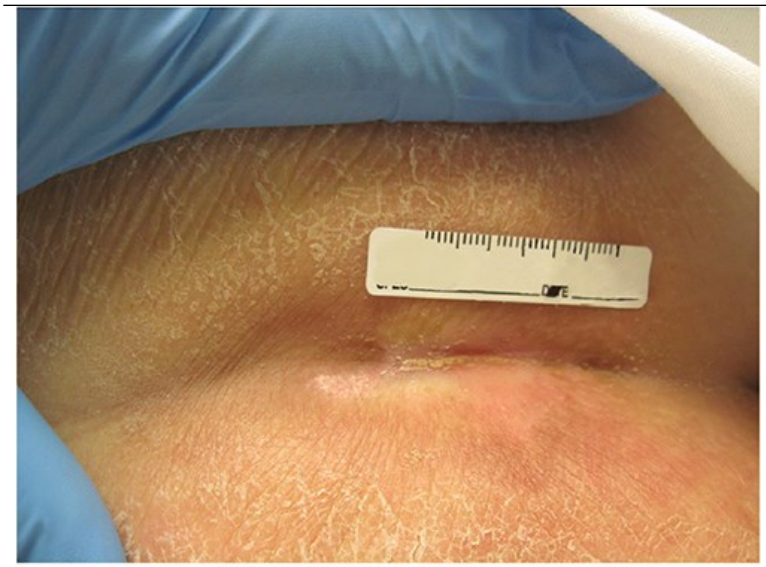

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At the time of the final visit, the wound is fully re-epithelialized. Nursing staff will continue to provide pressure redistribution in both sitting and supine positions and will cover the new fragile epithelial tissue with a foam dressing for protection from incontinence and friction. The patient will return to the clinic only if needed.

DISCUSSION QUESTIONS

1. What phase of healing is used to describe the wound in **FIGURE 12-51**?
2. What is the wound stage (using NPUAP Staging guidelines)?
3. Were all of the goals met?

FIGURE 12-41

Maggot debridement therapy: clinical series Dehisced transmetatarsal amputation before treatment with maggot therapy to debride the necrotic tissue.

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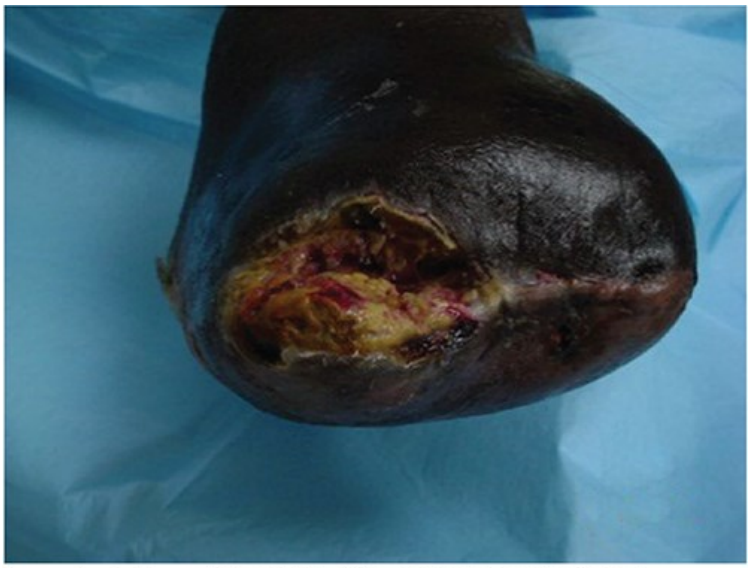

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FIGURE 12-42

Step 1 A barrier and "trough" made from hydrocolloid and hydrocolloid paste, onto which skin cement will be painted.

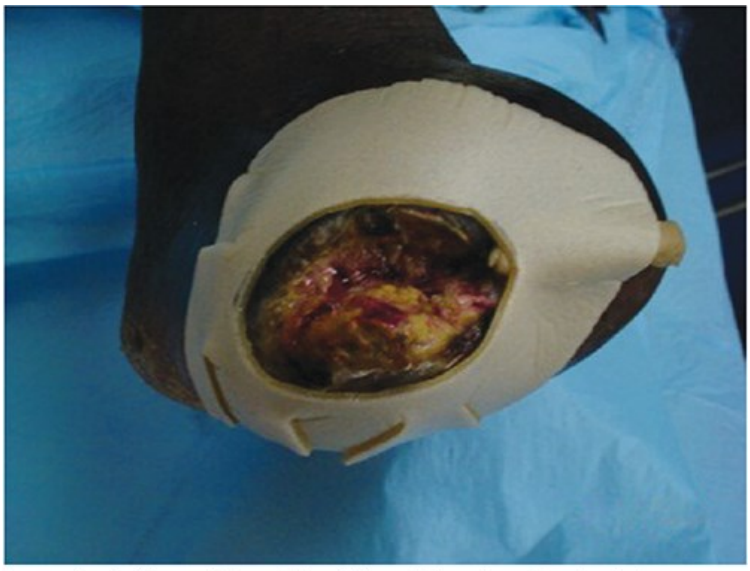

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FIGURE 12-43

Step 2 Wound veil preattached to barrier at the top, disinfected larvae applied using gauze containing nutrient that is included in the package with the larvae.

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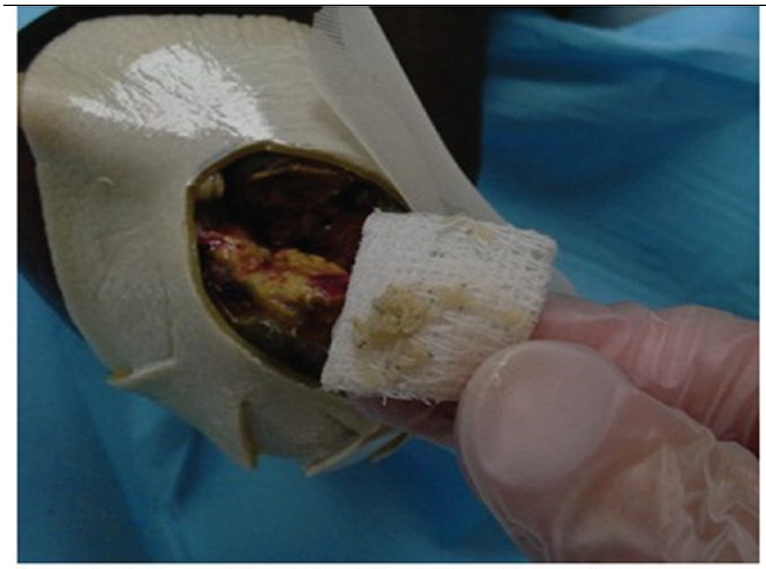

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FIGURE 12-44

Step 3 Larvae in place with veil attached to the hydrocolloid barrier and reinforced with waterproof tape.

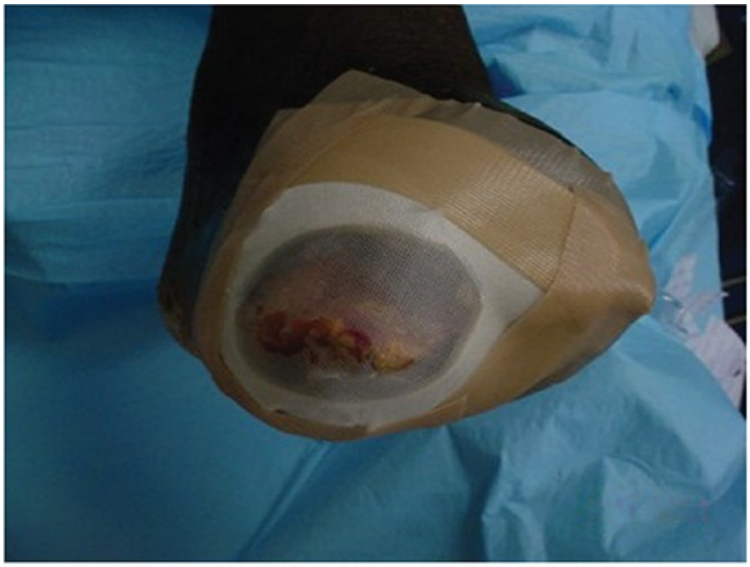

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FIGURE 12-45

Veil removed after 4–5 days of therapy to expose maggots Note the visible change in size after ingestion of necrotic debris.

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FIGURE 12-46

Wound after cleansing of slough and debris

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SUMMARY

Effective wound debridement is paramount to achieving adequate wound bed preparation leading to wound closure and subsequent healing, and has become a standard of care among wound care providers. Once the decision to debride is made, selection of the preferred method depends on the overall goals of therapy, the urgency of need, the setting in which the care is being provided, the availability of the debridement modalities, and the skill level of the care providers. Adequate wound debridement is essential to the realization of successful outcomes in the management of chronic wounds, and creating a clean wound environment free of necrotic tissue is essential prior to using modern, advanced wound therapies such as collagen dressings, growth factors, and tissue and cellular products.

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